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NEWS 21	Aug 19	The MEDLINE file segment of TOXCENTER has been reloaded
NEWS 22	Aug 26	Sequence searching in REGISTRY enhanced
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NEWS 27	Oct 21	EVENTLINE has been reloaded
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NEWS 29	Oct 24	Nutraceuticals International (NUTRACEUT) now available on STN
NEWS 30	Oct 25	MEDLINE SDI run of October 8, 2002
NEWS 31	Nov 18	DKILIT has been renamed APOLLIT
NEWS 32	Nov 25	More calculated properties added to REGISTRY
NEWS 33	Dec 02	TIBKAT will be removed from STN
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NEWS 36	Dec 17	TOXCENTER enhanced with additional content
NEWS 37	Dec 17	Adis Clinical Trials Insight now available on STN
NEWS 38	Dec 30	ISMEC no longer available
NEWS 39	Jan 13	Indexing added to some pre-1967 records in CA/CAPLUS
NEWS 40	Jan 21	NUTRACEUT offering one free connect hour in February 2003
NEWS 41	Jan 21	PHARMAML offering one free connect hour in February 2003
NEWS 42	Jan 29	Simultaneous left and right truncation added to COMPENDEX, ENERGY, INSPEC
NEWS 43	Feb 13	CANCERLIT is no longer being updated

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  NEWS EXPRESS  January 6 CURRENT WINDOWS VERSION IS V6.01a,
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=> s fusion protein
L1      138716 FUSION PROTEIN

=> s l1 and Fc fusion
L2        1701 L1 AND FC FUSION

=> s l2 and CD79
L3           0 L2 AND CD79

=> s CD79
L4         226 CD79

=> s l4 and fusion
L5           5 L4 AND FUSION

=> dup remove l5
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L6           5 DUP REMOVE L5 (0 DUPLICATES REMOVED)

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L6 ANSWER 1 OF 5 MEDLINE

2002645523 Document Number: 22271083. PubMed ID: 12384401. The alternative transcript of CD79b is overexpressed in B-CLL and inhibits signaling for apoptosis. Cragg Mark S; Chan H T Claude; Fox Mathew D; Tutt Alison; Smith Aimee; Oscier David G; Hamblin Terry J; Glennie Martin J. (Tenovus Research Laboratory, Cancer Sciences Division, University of Southampton School of Medicine, Southampton General Hospital, Tremona Road, Southampton SO16 6YD, UK.. msc@soton.ac.uk) . BLOOD, (2002 Nov 1) 100 (9) 3068-76. Journal code: 7603509. ISSN: 0006-4971. Pub. country: United States. Language: English.

AB The B-cell receptor (BCR) for antigen is composed of surface immunoglobulin (sIg), which provides antigen specificity, and a noncovalently associated signaling unit, the CD79a/b heterodimer. Defects in **CD79** can influence both BCR expression and signaling and may explain why cells from certain malignancies, such as B-chronic lymphocytic leukemia (B-CLL), often express diminished and inactive BCR. Recently, an alternative transcript of CD79b (DeltaCD79b) has been reported that is up-regulated in B-CLL and may explain this diminished BCR expression. Here we assess the expression of DeltaCD79b in B-CLL and other lymphoid malignancies and investigate its function. High relative expression of DeltaCD79b was confirmed in most cases of B-CLL and found in 6 of 6 cases of splenic lymphomas with villous lymphocytes (SLVLs) and hairy cell leukemia. In a range of Burkitt lymphoma cell lines, expression of DeltaCD79b was relatively low but correlated inversely with the ability of the BCR to signal apoptosis when cross-linked by antibody (Ab). Interestingly, when Ramos-EHRB cells, which express low DeltaCD79b, were transfected with this transcript, they were transformed from being sensitive to anti-Fcmu-induced apoptosis to being highly resistant. Although DeltaCD79b was expressed as protein, its overexpression did not reduce the level of cell surface BCR. Finally, we showed that the inhibitory activity of DeltaCD79b depended on an intact leader sequence to ensure endoplasmic reticulum (ER) trafficking and a functional signaling immunoreceptor tyrosine-based activation motif (ITAM) in its cytoplasmic tail. These results point to DeltaCD79b being a powerful modulator of BCR signaling that may play an important role in normal and malignant B cells.

L6 ANSWER 2 OF 5 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

2002:199074 Document No.: PREV200200199074. Gene expression profiling in two forms of acute leukemia involving the AML1 gene: Comparison to normal CD34+ progenitor cells. Hokland, Peter (1); Thykjaer, Thomas; Orntoft, Torben; Holm, Mette Skov (1); Pallisgaard, Niels (1). (1) Hematology, Aarhus University Hospital, Aarhus Denmark. Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 566a. http://www.bloodjournal.org/. print. Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001 ISSN: 0006-4971. Language: English.

AB Acute leukemias can be divided into lymphoid (ALL) and myeloid (AML) according to standard diagnostic criteria, most notably immunophenotyping. However, underlying this classification is a remarkable heterogeneity, as evidenced by the increasing number of recurrent chromosome abnormalities found in these disorders, especially balanced translocations. Moreover, despite a widening knowledge of the genes involved in such genetic lesions, little is known about leukemic processes. We have employed oligonucleotide-based DNA microarrays to study the global gene expression of more than 6600 genes in 3 cases of TEL/AML+ (t(12;21)) pre-B ALL and in 3 cases of AML/ETO+ (t(8;21)) AML. When gene expression was compared in these two disorders, which both involve the AML gene on chromosome 21, we found them to be clearly distinct from each other and from mobilized CD34+ cells from normal donors, with AML/ETO+ cases resembling normal CD34+ more than the TEL/AML ones. In the TEL/AML+ patients, 165 genes were either more than 3-fold increased or decreased compared to normal CD34+ cells. For ETO/AML+ patients the number was 93. A striking homogeneity within the

patient groups and the normal CD34+ cells was common to all gene expression alterations, probably reflecting the fact that focused subsets within AML and ALL patients were chosen a priori. Categorizing genes according to cellular function revealed that a number of genes already known to exert important functions in either lymphoid or myeloid cells were differentially expressed. Thus, while such genes as CD10, CD19, CD79alpha (MB-1) and IL-7R were more than 10-fold overexpressed in TEL/AML+ cases, a range of enzymes related to myeloid cell function such as myeloperoxidase, elastase and proteinase-3 were all more than 20-fold overexpressed in AML/ETO. More importantly, however, was the unexpected finding that some heat-shock proteins were clearly downregulated in both TEL/AML and AML/ETO, most notably hsp40 (8 and 30 fold, respectively) and hsp70 (59 and 47 fold, respectively). Interestingly, hsp27, which has previously been found to be upregulated in some forms of pre-B ALL, was unaltered in both patient groups. In addition, a series of transcription factors were differentially regulated between the subgroups, while a group of cell cycle related genes were upregulated in AML/ETO+ cells compared to both TEL/AML+ and normal CD34+ cells. Finally, 11 number genes with unknown function (ESTs) were been identified. The magnitude of gene expression alterations identified by the chip methodology was validated in separate patients employing TaqMan real-time quantitative PCR with the exception of CD10 (CALLA), which was found to be 100-fold more expressed in the RQ-PCR assay. We conclude that focused gene expression profiling is a valuable tool to distinguish subsets of acute leukemias already characterized by distinct chromosomal aberrations. Moreover, even subsets with balanced translocations studied here, which share one **fusion** partner, display remarkably dissimilar gene profiles. These data therefore form the basis for further investigations into the leukemogenesis and for refining the diagnostic processes in these diseases.

L6 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2003 ACS

2000:756855 Document No. 133:318803 Synthetic signal transducing proteins using motifs associated with receptor binding and activation. Lawson, Alastair David Griffiths; Finney, Helene Margaret (Celltech Therapeutics Limited, UK). PCT Int. Appl. WO 2000063372 A1 20001026, 74 pp. DESIGNATED STATES: W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 2000-GB1456 20000417. PRIORITY: GB 1999-8807 19990416.

AB The invention relates to synthetic signalling mols., which are based on sequences derived from primary signalling motifs such as Ig tyrosine receptor-based activation motifs (ITAMs). The use of such signalling mols. within chimeric receptor proteins allows one to tailor the level of intracellular signalling mediated by the chimeric receptor. Proteins contg., and nucleic acids encoding, such synthetic signalling mols. suitable for use in medicine, are described.

L6 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2003 ACS

1999:795994 Document No. 132:31744 Gene probes used for genetic profiling in healthcare screening and planning. Roberts, Gareth Wyn (Genostic Pharma Ltd., UK). PCT Int. Appl. WO 9964627 A2 19991216, 745 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2.

APPLICATION: WO 1999-GB1780 19990604. PRIORITY: GB 1998-12099 19980606; GB 1998-13291 19980620; GB 1998-13611 19980624; GB 1998-13835 19980627; GB 1998-14110 19980701; GB 1998-14580 19980707; GB 1998-15438 19980716; GB 1998-15576 19980718; GB 1998-15574 19980718; GB 1998-16085 19980724; GB 1998-16086 19980724; GB 1998-16921 19980805; GB 1998-17097 19980807; GB 1998-17200 19980808; GB 1998-17632 19980814; GB 1998-17943 19980819.

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of genes enables the invention of a design for genetic profiling technologies which comprises of the identification of the core group of genes and their sequence variants required to provide a broad base of clin. prognostic information - "genostics". The "Genostic" profiling of patients and persons will radically enhance the ability of clinicians, healthcare professionals and other parties to plan and manage healthcare provision and the targeting of appropriate healthcare resources to those deemed most in need. The use of this invention could also lead to a host of new applications for such profiling technologies, such as identification of persons with particular work or environment related risk, selection of applicants for employment, training or specific opportunities or for the enhancing of the planning and organization of health services, education services and social services.

L6 ANSWER 5 OF 5 CAPLUS COPYRIGHT 2003 ACS

1999:795993 Document No. 132:31743 Gene probes used for genetic profiling in healthcare screening and planning. Roberts, Gareth Wyn (Genostic Pharma Limited, UK). PCT Int. Appl. WO 9964626 A2 19991216, 149 pp. DESIGNATED STATES: W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG. (English). CODEN: PIXXD2. APPLICATION: WO 1999-GB1779 19990604. PRIORITY: GB 1998-12098 19980606; GB 1998-28289 19981223.

AB There is considerable evidence that significant factor underlying the individual variability in response to disease, therapy and prognosis lies in a person's genetic make-up. There have been numerous examples relating that polymorphisms within a given gene can alter the functionality of the protein encoded by that gene thus leading to a variable physiol. response. In order to bring about the integration of genomics into medical practice and enable design and building of a technol. platform which will enable the everyday practice of mol. medicine a way must be invented for the DNA sequence data to be aligned with the identification of genes central to the induction, development, progression and outcome of disease or physiol. states of interest. According to the invention, the no. of genes and their configurations (mutations and polymorphisms) needed to be identified in order to provide crit. clin. information concerning individual prognosis is considerably less than the 100,000 thought to comprise the human genome. The identification of the identity of the core group of

genes enables the invention of a design for genetic profiling technologies.

=> s l4 and CD79alpha
L7 6 L4 AND CD79ALPHA

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PROCESSING COMPLETED FOR L7
L8 6 DUP REMOVE L7 (0 DUPLICATES REMOVED)

=> d l8 1-6 cbib abs

L8 ANSWER 1 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
2002:119309 Document No.: PREV200200119309. Follicular Hodgkin lymphoma: A histopathologic study. Kansal, Rina; Singleton, Timothy P.; Ross, Charles W.; Finn, William G.; Padmore, Ruth F.; Schnitzer, Bertram (1). (1) Dept of Pathology, University of Michigan Medical Center, 1301 Catherine Rd, Ann Arbor, MI, 48109-0602 USA. American Journal of Clinical Pathology, (January, 2002) Vol. 117, No. 1, pp. 29-35. http://www.ajcp.com. print. ISSN: 0002-9173. Language: English.
AB Follicular Hodgkin lymphoma (FHL) is a form of classic Hodgkin lymphoma with morphologic similarity to nodular lymphocyte predominant Hodgkin lymphoma (NLPHL). We present the clinicopathologic features of 13 FHL cases and compare their morphologic features with 40 cases of NLPHL. Seven males and 6 females had FHL in the lymph nodes of the neck (6 patients), axilla (3 patients), groin (2 patients), and mediastinum (1 patient) and in the nasopharynx (1 patient). All FHLs had follicles with small, eccentric germinal centers (GCs) and expanded mantle zones containing classic Reed-Sternberg (RS) cells; reactive GCs also were seen in 6 of 13 cases. The RS cells were CD30+, fascin+ in 13 cases; CD15+ in 11 cases; CD20+ in 4 cases; **CD79alpha**+CD20- in 1 case; and negative for epithelial membrane antigen in 12 cases; they were surrounded by CD3epsilon+ and CD57+ rosettes in 13 and 2 cases, respectively. Morphologically, FHL may closely simulate NLPHL, and, thus, immunohistochemical analysis is essential to confirm the diagnosis. Clues helpful in diagnosing FHL include the presence of follicles with GCs, classic RS cells, and a relative absence of histiocytes.

L8 ANSWER 2 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
2001:333020 Document No.: PREV200100333020. Plasmacytoma with aberrant expression of myeloid markers, T-cell markers, and cytokeratin. Shin, Jung S.; Stopyra, Gary A.; Warhol, Michael J.; Multhaupt, Hinke A. B. (1). (1) Dept. of Pathology, Pennsylvania Hospital, 800 Spruce Street, Philadelphia, PA, 19107: himult@pahosp.com USA. Journal of Histochemistry and Cytochemistry, (June, 2001) Vol. 49, No. 6, pp. 791-792. print. ISSN: 0022-1554. Language: English. Summary Language: English.
AB Plasmacytomas are localized neoplastic proliferations of monoclonal plasma cells. When multifocal, the process is referred to as multiple myeloma. These lesions exhibit a pattern of antigen expression and cytomorphology that usually leads to a ready diagnosis. However, potentially troublesome variations in immunophenotype occur. We describe a case of a plasmacytoma from a patient who presented with sudden onset of pain and a lytic lesion of the left proximal humerus. Hematoxylin and eosin-stained sections showed a lymphoproliferative lesion composed of large lymphoid cells, some with plasmacytoid and immunoblastic features. The lesion also showed significant mitotic activity. Immunohistochemical staining was positive for CD45 (LCA), CD56 (N-CAM), CD43 (MT1), and cytokeratin CAM5.2. There was also clonal staining for lambda light chains. In addition, flow cytometric analysis showed positivity for myeloid markers such as CD13, CD33, CD38, and CD138. Significant negative markers include CD20 (L26), CD45RO (UCHL-1), and **CD79alpha**. The unusual phenotypic features of this plasmacytoma illustrate potential diagnostic pitfalls. It is

important to fully study such lesions to correctly classify them, because this has significant impact on prognosis and management.

L8 ANSWER 3 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
2002:199074 Document No.: PREV200200199074. Gene expression profiling in two forms of acute leukemia involving the AML1 gene: Comparison to normal CD34+ progenitor cells. Hokland, Peter (1); Thykjaer, Thomas; Orntoft, Torben; Holm, Mette Skov (1); Pallisgaard, Niels (1). (1) Hematology, Aarhus University Hospital, Aarhus Denmark. Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 566a. http://www.bloodjournal.org/. print. Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001 ISSN: 0006-4971. Language: English.

AB Acute leukemias can be divided into lymphoid (ALL) and myeloid (AML) according to standard diagnostic criteria, most notably immunophenotyping. However, underlying this classification is a remarkable heterogeneity, as evidenced by the increasing number of recurrent chromosome abnormalities found in these disorders, especially balanced translocations. Moreover, despite a widening knowledge of the genes involved in such genetic lesions, little is known about leukemic processes. We have employed oligonucleotide-based DNA microarrays to study the global gene expression of more than 6600 genes in 3 cases of TEL/AML+ (t(12;21)) pre-B ALL and in 3 cases of AML/ETO+ (t(8;21)) AML. When gene expression was compared in these two disorders, which both involve the AML gene on chromosome 21, we found them to be clearly distinct from each other and from mobilized CD34+ cells from normal donors, with AML/ETO+ cases resembling normal CD34+ more than the TEL/AML ones. In the TEL/AML+ patients, 165 genes were either more than 3-fold increased or decreased compared to normal CD34+ cells. For ETO/AML+ patients the number was 93. A striking homogeneity within the patient groups and the normal CD34+ cells was common to all gene expression alterations, probably reflecting the fact that focused subsets within AML and ALL patients were chosen a priori. Categorizing genes according to cellular function revealed that a number of genes already known to exert important functions in either lymphoid or myeloid cells were differentially expressed. Thus, while such genes as CD10, CD19, **CD79alpha** (MB-1) and IL-7R were more than 10-fold overexpressed in TEL/AML+ cases, a range of enzymes related to myeloid cell function such as myeloperoxidase, elastase and proteinase-3 were all more than 20-fold overexpressed in AML/ETO. More importantly, however, was the unexpected finding that some heat-shock proteins were clearly downregulated in both TEL/AML and AML/ETO, most notably hsp40 (8 and 30 fold, respectively) and hsp70 (59 and 47 fold, respectively). Interestingly, hsp27, which has previously been found to be upregulated in some forms of pre-B ALL, was unaltered in both patient groups. In addition, a series of transcription factors were differentially regulated between the subgroups, while a group of cell cycle related genes were upregulated in AML/ETO+ cells compared to both TEL/AML+ and normal CD34+ cells. Finally, 11 number genes with unknown function (ESTs) were been identified. The magnitude of gene expression alterations identified by the chip methodology was validated in separate patients employing TaqMan real-time quantitative PCR with the exception of CD10 (CALLA), which was found to be 100-fold more expressed in the RQ-PCR assay. We conclude that focused gene expression profiling is a valuable tool to distinguish subsets of acute leukemias already characterized by distinct chromosomal aberrations. Moreover, even subsets with balanced translocations studied here, which share one fusion partner, display remarkably dissimilar gene profiles. These data therefore form the basis for further investigations into the leukemogenesis and for refining the diagnostic processes in these diseases.

L8 ANSWER 4 OF 6 EMBASE COPYRIGHT 2003 ELSEVIER SCI. B.V.
2001345804 EMBASE Immunohistological study of the immune system cells in paraffin-embedded tissues of conventional pigs. Chianini F.; Majo N.; Segales J.; Dominguez J.; Domingo M.. F. Chianini, Dept. de Sanitat i

Anatomia Animals, Ctr. Recerca Sanitat Animal (CReSA), Univ. Auton. Barcelona, Bellaterra, 08193 Barcelona, Spain. fchianini@blues.uab.es.
Veterinary Immunology and Immunopathology 82/3-4 (245-255) 2001.
Refs: 20.
ISSN: 0165-2427. CODEN: VIIMDS.
Publisher Ident.: S 0165-2427(01)00364-6. Pub. Country: Netherlands.
Language: English. Summary Language: English.

AB The distribution of different cells of the immune system has been studied in formalin-fixed paraffin-embedded tissues from conventionally reared healthy pigs, using immunohistological techniques. The samples collected were: lungs, tonsils, lymph nodes (mediastinal, mesenteric, inguinal and submandibular), pancreas, spleen, liver, kidney, adrenal gland, ileum and stomach. A total of six primary antibodies anti-CD3, anti-**CD79** .alpha., Mac 387, anti-lysozyme, anti-CD45RA (3C3/9) and anti-SLA-II-DQ (BL2H5) were used with a standard avidin-biotin peroxidase (ABC) method. Anti-CD3 and anti-**CD79**.alpha. mAb-reacted, respectively with cells located in T cell areas and B cell areas. Mac 387 recognised circulating polymorphonuclear leukocytes, while anti-lysozyme-stained resident macrophages in all tissues. 3C3/9 and BL2H5, were assessed in formalin-fixed paraffin-embedded tissues for the first time. 3C3/9 identified B lymphocytes, in primary follicles and mantle zones, a subpopulation of T cells, especially located in the marginal zone of the spleen and a variable number of immunoblasts, in the germinal centres. BL2H5 reacted with B cells in the mantle zones of the follicles of lymphoid tissues, with dendritic and interdigitating cells in all studied lymphoid tissues and with a variable number of resting and activated T cells in the periarteriolar lymphoid sheath (PALs), marginal zone and red pulp of the spleen. Furthermore, it stained Kupffer and perivascular macrophages in the liver This study represents a detailed histological study of the distribution of the most important subpopulations of immune system cells in conventional, healthy pigs. In our view, these tools should be useful for future comparative studies in disease conditions.

L8 ANSWER 5 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
2000:82688 Document No.: PREV200000082688. The role of the **CD79alpha** /CD79beta heterodimer and of the CD19/CD21/CD81 complex in the development and biologic function of B cell antigen receptor. Rupniewska, Zofia Monika (1); Dmoszynska, Magdalena (1). (1) ul. Jaczewskiego 8, 20-950, Lublin Poland. Postepy Biologii Komorki, (1999) Vol. 26, No. 4, pp. 841-861.
ISSN: 0324-833X. Language: Polish. Summary Language: English; Polish.

AB The B-cell receptor complex (BCR) is a multimeric complex which consists of a membrane immunoglobulin (mIg) molecule and the non-covalently associated **CD79alpha**/CD79beta heterodimer. It has been shown that this heterodimer is both essential and sufficient for the surface expression of mIg. Cytoplasmic tails of **CD79alpha** and CD79beta have a signaling capacity. CD19 is a B cell-restricted membrane protein of the immunoglobulin superfamily that associates with the BCR. Coligating globulin CD19 to mIgM reduces by 100-fold the threshold number of mIgM molecules that must be ligated to induce synthesis of DNA. CD19 is a core member of a multimolecular cell surface signal-transduction complex, with contains four identified proteins including CD19, CD21 (complement receptor type 2), CD81 (tetraspan family of membrane proteins), and Leu-13 as well as other unidentified proteins. The association between CD19 and CD21 provides mechanism for binding the CD19 complex with the BCR through complexes Ag and C3 fragments. This is likely to have important consequences for the development of natural and acquired immunity during B cell development and immune responses.

L8 ANSWER 6 OF 6 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
1998:48155 Document No.: PREV199800048155. **CD79alpha**/CD79beta heterodimers are expressed on pro-B cell surfaces without associated mu heavy chain. Koyama, Mariko; Ishihara, Katsuhiko; Karasuyama, Hajime;

Cordell, Jacqueline L.; Iwamoto, Aikichi; Nakamura, Tetsuya (1). (1) Dep. Infect. Dis. Applied Immunol., Inst. Med. Sci., Univ. Tokyo, 4-6-1 Shirokanedai, Minato-ku, Tokyo 108 Japan. International Immunology, (Nov., 1997) Vol. 9, No. 11, pp. 1767-1772. ISSN: 0953-8178. Language: English.

AB During B cell development, the surface expression of **CD79alpha** /CD79beta heterodimers had been thought to begin in the pre-B cell stage where the heterodimers constitute pre-B cell receptors together with mu heavy and surrogate light chains. Thereafter, in mature B cells, **CD79alpha**/CD79beta associates with surface Ig to form B cell antigen receptors. In this study, we revealed by using newly established mAb that CD79beta was expressed on the surface of pro-B cells which had not undergone the productive Ig gene rearrangement. Biochemical analysis showed that CD79beta on pro-B cells existed either as monomers or as disulfide-linked heterodimers with **CD79alpha**, non-covalently associated with four unidentified membrane molecules. Our finding that CD79beta is expressed on earlier B-lineage cells than previously expected coincides with the recent study in which CD79beta-deficient mice exhibit a blockade of B cell differentiation at the pro-B cell stage. Thus, it is speculated that the CD79beta-containing complexes on pro-B cell surfaces may function to induce early B cell differentiation.

=> s (chang t?/au or sheu j?/au or huang j?/au or wu s?/au or chen l?/au)
L9 91121 (CHANG T?/AU OR SHEU J?/AU OR HUANG J?/AU OR WU S?/AU OR CHEN L?/AU)

=> s l9 and CD79
L10 0 L9 AND CD79

=> s l9 and Fc fusion
L11 19 L9 AND FC FUSION

=> dup remove l11
PROCESSING COMPLETED FOR L11
L12 7 DUP REMOVE L11 (12 DUPLICATES REMOVED)

=> d l12 1-7 cbib abs

L12 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2003 ACS
2003:97801 Treatment of tumors and viral infections with a hybrid conjugate of interferon and an immunoglobulin fc. Yu, Liming; **Chang, Tse Wen** (USA). U.S. Pat. Appl. Publ. US 20030026779 A1 20030206, 9 pp., Cont. of U.S. Ser. No. 418,734, abandoned. (English). CODEN: USXXCO. APPLICATION: US 2001-5438 20011203. PRIORITY: US 1999-418734 19991015.

AB The present invention relates to interferon-immunoglobulin **Fc fusion** proteins (referred to as "IFN-Fc hybrids") and their use in treating tumors. The IFN-Fc hybrids preferably (but not necessarily) include linkers between the IFN and the Fc portion, and the IFN portion can be an IFN variant. These linkers are preferably composed of a T cell inert sequence, or any non-immunogenic sequence, including Gly-Ser repeat units. The preferred Fe fragment is a human immunoglobulin Fc fragment, preferably the .gamma.4 chain.

L12 ANSWER 2 OF 7 MEDLINE DUPLICATE 1
2003031355 Document Number: 22426461. PubMed ID: 12538671. Comparative effects of human igalpha and igbeta in inducing autoreactive antibodies against B cells in mice. **Sheu Jim J C**; Cheng Tammy; Chen Huan Y; Lim Carmay; **Chang Tse-Wen**. (Departments of. Life Science and. Chemistry, National Tsing Hua University, Hsinchu, Taiwan. Institute of Biomedical Science, Academia Sinica, Taipei, Taiwan. Development Center for Biotechnology, Taipei, Taiwan.) JOURNAL OF IMMUNOLOGY, (2003 Feb 1) 170 (3) 1158-66. Journal code: 2985117R. ISSN: 0022-1767. Pub. country: United States. Language: English.

AB Human and mouse Igalpha molecules share only 58% amino acid sequence identity in their extracellular regions. However, mice immunized with a recombinant **Fc fusion** protein containing the extracellular portion of human Igalpha produced significant amounts of IgG capable of binding to Igalpha on mouse B cells. The induced auto/cross-reactive Abs could down-regulate B cell levels and the consequent humoral immune responses against an irrelevant Ag in treated mice. Analogous immunization with an **Fc fusion** protein containing the extracellular portion of human Igbeta gave a much weaker response to mouse Igbeta, although human and mouse Igbeta, like their Igalpha counterparts, share 56% sequence identity in their extracellular regions. Protein sequence analyses indicated that a potential immunogenic segment, located at the C-terminal loop of the extracellular domain, has an amino acid sequence that is identical between human and mouse Igalpha. A mAb A01, which could bind to both human and mouse Igalpha, was found to be specific to a peptide encompassing this immunogenic segment. These findings suggest that specific auto/cross-reactivity against self Igalpha can be induced by a molecular mimicry presented by a foreign Igalpha.

L12 ANSWER 3 OF 7 MEDLINE DUPLICATE 2

2002459345 Document Number: 22206420. PubMed ID: 12218370. Monoclonal antibodies against the C(epsilon)mX domain of human membrane-bound IgE and their potential use for targeting IgE-expressing B cells. Chen Huan Yuan; Liu Fu-Tong; Hou Charlie M H; **Huang Janice S W**; Sharma Bhavya Bhavna; **Chang Tse Wen**. (Department of Life Science, National Tsing Hua University, Hsinchu, Taiwan, ROC.) INTERNATIONAL ARCHIVES OF ALLERGY AND IMMUNOLOGY, (2002 Aug) 128 (4) 315-24. Journal code: 9211652. ISSN: 1018-2438. Pub. country: Switzerland. Language: English.

AB BACKGROUND: IgE mediates immediate-type hypersensitivity reactions responsible for various allergic symptoms. It is secreted by IgE-producing plasma cells, which differentiate from B cells expressing membrane-bound IgE (mIgE) on their surface. The epsilon-chain of human mIgE contains a membrane-anchoring peptide and an extra 52-amino-acid (a.a.)-long domain (referred to as C(epsilon)mX) between the membrane anchor and the CH4 domain. OBJECTIVE: The study was designed to evaluate the effects of C(epsilon)mX-specific monoclonal antibodies (mAbs) to target IgE-expressing B cells and decrease IgE production. METHODS: A C(epsilon)mX-containing IgG1.**Fc fusion** protein was produced in CHO cells and used to immunize mice; five hybridoma clones secreting C(epsilon)mX-specific mAbs were obtained. RESULTS: Characterization of the mAbs using ELISA, immunoprecipitation, and immunoblotting methods showed that they could bind to both native and denatured forms of C(epsilon)mX. The mAbs exhibited mutual inhibition of binding to mIgE. Epitope mapping using synthetic peptides revealed that all five mAbs recognize the same epitope, RADWPGPP, located near the C-terminus of C(epsilon)mX. Binding of one of the mAbs to mIgE on SKO-007 cells induced the cross-linking of mIgE molecules on the cell surface, resulting in their patching and capping. In vitro functional analysis revealed that mAbs are able to cause complement-mediated cytotoxicity on transfectants expressing the Fc portion of mIgE. CONCLUSION: We have prepared several human mIgE-specific mAbs. The potential of the mAbs on targeting mIgE+ B cells was demonstrated by CDC analysis.

L12 ANSWER 4 OF 7 MEDLINE DUPLICATE 3

2002207068 Document Number: 21937790. PubMed ID: 11941453. Inducing specific reactivity against B cells in mice by immunizing with an **Fc fusion** protein containing self-Igbeta. **Sheu Jim J C; Huang Janice; Chang Tse W**. (Department of Life Science, National Tsing Hua University, Hsinchu 300, Taiwan.) CANCER IMMUNOLOGY, IMMUNOTHERAPY, (2002 May) 51 (3) 145-52. Journal code: 8605732. ISSN: 0340-7004. Pub. country: Germany: Germany, Federal Republic of. Language: English.

AB A recombinant chimeric fusion protein, muIgbeta-hugamma4.Fc, composed of the extracellular domain of mouse Igbeta (CD79b) and the CH2-CH3 domains of human IgGgamma4.Fc (hugamma4.Fc), linked via an immunologically inert flexible peptide, was prepared. The fusion protein was evaluated for its ability to induce specific auto-reactive immune response against Igbeta and to modulate B cell activity in Balb/c mice. Upon immunization with muIgbeta-hugamma4.Fc, mice developed immunoglobulin (IgG) against self-Igbeta, which could bind to the cells of a mouse B cell line expressing Igbeta on the cell surface. The proportion of B cells in mononuclear cells in the peripheral blood (PBMC) of treated mice decreased as compared to that of mice immunized with hugamma4.Fc without the Igbeta component. Furthermore, mice immunized against muIgbeta-hugamma4.Fc displayed a reduced antibody response against an irrelevant antigen. The implications of employing the present approach in developing a therapeutic strategy for regulating B cell activity has been discussed.

L12 ANSWER 5 OF 7 MEDLINE DUPLICATE 4
2002140847 Document Number: 21830650. PubMed ID: 11841845. Down regulation of B cells by immunization with a fusion protein of a self CD20 peptide and a foreign IgG.Fc fragment. **Huang Janice; Sheu Jim Jinn Chyuan; Wu Stanley Chi Shen; Chang Tse Wen**. (College of Life Science, National Tsing Hua University, Hsinchu, Taiwan, ROC.) IMMUNOLOGY LETTERS, (2002 Apr 1) 81 (1) 49-58. Journal code: 7910006. ISSN: 0165-2478. Pub. country: Netherlands. Language: English.

AB In vivo studies of mice were performed to investigate whether auto-reactive antibodies specific for self CD20 antigen on B cells could be induced by immunizing with a CD20 peptide linked to a foreign, human IgG.Fc fragment through a T cell immunologically inert linker peptide and how such an auto-reactivity, if generated, would affect the levels of B cells. The dimeric **Fc fusion** protein containing the extracellular 44-amino acid portion of CD20, and the CH2-CH3 domains of human gamma 1 immunoglobulin were prepared. After several subcutaneous immunizations with this CD20-Fc protein, mice produced anti-CD20 antibodies that can bind to native CD20 on normal B cells and B-lymphoma cells. In mice immunized with the CD20-Fc protein, the fraction of B cells in total peripheral blood lymphocytes decreased to about 40%, significantly lower than that of mice immunized with human IgG. In addition, antibody response towards an irrelevant bystander antigen, chicken ovalbumin, was weakened compared with that of mice immunized with human IgG. These results show that auto-reactive antibodies specific for CD20 can be induced by immunizing with an autologous CD20 peptide fused with a foreign IgG.Fc and that the auto-antibodies can partially reduce the levels of B cells and their response to other antigens.

L12 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2003 ACS
1999:354301 Document No. 131:4242 Interferon-.beta. fusion protein with immunoglobulin Fc fragment. **Chang, Tse Wen;** Yu, Liming (Tanox, Inc., USA). U.S. US 5908626 A 19990601, 7 pp., Cont.-in-part of U.S. 5,723,125. (English). CODEN: USXXAM. APPLICATION: US 1997-994719 19971219. PRIORITY: US 1995-579211 19951228; US 1996-719331 19960925.

AB The authors disclose a hybrid recombinant protein consisting of human interferon-.beta. and an Ig Fc fragment (preferably .gamma.4 chain) joined by a peptide linker. The hybrid mol. has increased circulatory half-life.

L12 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2003 ACS
1998:157344 Document No. 128:216372 Hybrid with interferon-alpha and an immunoglobulin Fc linked through a non-immunogenic peptide. **Chang, Tse Wen;** Yu, Liming (Tanox Biosystems, Inc., USA). U.S. US 5723125 A 19980303, 8 pp., Cont.-in-part of U.S. Ser. No. 579,211, abandoned. (English). CODEN: USXXAM. APPLICATION: US 1996-719331 19960925. PRIORITY: US 1995-579211 19951228.

AB Disclosed is a hybrid recombinant protein consisting of human interferon,

preferably interferon-.alpha. (IFN.alpha.), and human Ig Fc fragment, preferably .gamma.4 chain, joined by a peptide linker comprising the sequence Gly Gly Ser Gly Gly Ser Gly Gly Gly Gly Ser Gly Gly Gly Gly Ser (SEQ ID NO:1). Plasmid pCDNA3/IFN.alpha.-Fc encoding the disclosed chimeric interferon-.alpha./Ig .gamma.4 was prepd. The chimeric interferon .alpha. is useful for treating inflammatory, viral and malignant diseases.

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=> s CD79-Fc
L13             0 CD79-FC

=> s CD79 hybrid
L14             0 CD79 HYBRID

=> d his

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     13 FEB 2003
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L2           1701 S L1 AND FC FUSION
L3              0 S L2 AND CD79
L4            226 S CD79
L5              5 S L4 AND FUSION
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L7              6 S L4 AND CD79ALPHA
L8              6 DUP REMOVE L7 (0 DUPLICATES REMOVED)
L9          91121 S (CHANG T?/AU OR SHEU J?/AU OR HUANG J?/AU OR WU S?/AU OR CHEN
L10             0 S L9 AND CD79
L11            19 S L9 AND FC FUSION
L12             7 DUP REMOVE L11 (12 DUPLICATES REMOVED)
L13             0 S CD79-FC
L14             0 S CD79 HYBRID

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=> s l15 and chimer
L17             0 L15 AND CHIMER

=> s l15 and Fc
L18             3 L15 AND FC

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L19             3 DUP REMOVE L18 (0 DUPLICATES REMOVED)

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L19  ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2003 ACS
2001:489619   Document No. 135:71268  Use of locked nucleic acid-modified
     oligonucleotides for treatment of cancer and inflammation.  Orum, Henrik;
     Koch, Troel; Skouv, Jan; Jakobsen, Mogen Havsteen (Exiqon A/S, Den.).  PCT
     Int. Appl. WO 2001048190 A2 20010705, 50 pp.  DESIGNATED STATES: W:  AE,
     AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CR, CU, CZ,
     DE, DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS,
     JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN,
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MW, MX, MZ, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM; RW: AT, BE, BF, BJ, CF, CG, CH, CI, CM, CY, DE, DK, ES, FI, FR, GA, GB, GR, IE, IT, LU, MC, ML, MR, NE, NL, PT, SE, SN, TD, TG, TR. (English). CODEN: PIXXD2. APPLICATION: WO 2000-IB2043 20001222. PRIORITY: US 1999-PV171873 19991223.

AB The invention relates to therapeutic applications of LNA-modified oligonucleotides. In particular, the invention provides methods for treatment of undesired cell growth as well as treatment of inflammatory related diseases and disorders. Preferably, administration of an LNA-modified oligonucleotide modulates expression of a targeted gene assocd. with the undesired cell growth or an inflammatory related disease or disorder. Thus, the peritoneal cells of rats injected i.p. with LNA-contg. oligonucleotides directed to **Fc**.epsilon.R1.alpha. mRNA produced less **Fc**.epsilon.R1.alpha. and released less histamine than did rats given unmodified oligonucleotides.

L19 ANSWER 2 OF 3 MEDLINE

1998023469 Document Number: 98023469. PubMed ID: 9358611. The development of anti-**CD79** monoclonal antibodies for treatment of B-cell neoplastic disease. Zhang L; French R R; Chan H T; O'Keefe T L; Cragg M S; Power M J; Glennie M J. (Lymphoma Research Unit, Tenovus Laboratory, General Hospital, Southampton, UK.) THERAPEUTIC IMMUNOLOGY, (1995 Aug) 2 (4) 191-202. Journal code: 9421528. ISSN: 0967-0149. Pub. country: ENGLAND: United Kingdom. Language: English.

AB The B-cell antigen receptor (BCR) consists of cell surface IgM associated with the **CD79** alpha/beta heterodimer. In this paper we describe a panel of monoclonal antibodies (mAbs) recognising the extracellular regions of human **CD79** alpha and beta. FACS analysis demonstrated that the mAbs bind to a range of Burkitt's lymphoma lines, a mouse B-cell line (JO-72) transfected with human **CD79** alpha and beta, and tumour biopsies from NHL patients. The specificity of the mAbs was confirmed by immunoprecipitation. The Ka for the binding of IgG from the anti-**CD79** alpha mAbs to cell surface **CD79** alpha on Ramos cells was 3 x 10(8) M-1, and their maximum level of binding, 1.7-2 x 10(5) molecules/cell, matched that obtained with anti-**Fc** mu and anti-Fd mu mAbs. All four anti-**CD79** beta mAbs were of lower affinity. Interestingly, in growth arrest studies, we found that while all anti-**Fc** mu mAbs caused profound inhibition of proliferation of Ramos cells, a range of other anti-BCR mAbs, which included the anti-**CD79**, anti-Fab mu, anti-gamma and anti-idiotype reagents, all performed poorly giving a maximum of 25% inhibition. These differences in performance are believed to relate to the ability of anti-BCR mAbs to cross-link neighbouring surface BCR and suggest that, unlike anti-**Fc** mu which favours cross-linking, most of these mAbs are binding in a monogamous, non-cross-linking, union with the BCR.

L19 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2003 ACS

1993:558224 Document No. 119:158224 Fluorescent monoclonal antibodies for flow cytometric classification and monitoring of leukemias. Terstappen, Leon W. M. M. (Becton, Dickinson and Co., USA). U.S. US 5234816 A 19930810, 36 pp. (English). CODEN: USXXAM. APPLICATION: US 1991-731217 19910712.

AB Leukocytes from leukemia patients are classified as to leukemic type by (a) dividing each sample of leukocytes into aliquots, (b) mixing the aliquots with different pairs of monoclonal antibodies (where each antibody in a pair is labeled with a different fluorochrome), (c) analyzing the cells in each aliquot for light scatter and fluorescence by flow cytometry, (d) constructing a log-log plot of fluorescence emission of each cell for the 2 fluorochromes, (e) dividing each plot into quadrants corresponding to double pos., double neg., and single pos. for each antibody, (f) numbering the quadrants of each plot consecutively, (g) assigning to each aliquot quadrant nos. for the quadrant(s) wherein the

percentage of pos. cells exceeds a threshold no. (e.g. 20% or 30%), and (h) comparing the quadrant patterns of the aliquots with those of known leukemia types. Treatment may be monitored by comparing the scores before, during, and after treatment. Thus, leukemia patients were classified as acute B-lymphoid, acute T-lymphoid, or acute myeloid based on patterns of leukocyte staining with CD10/CD19, CD20/CD5, CD3/CD22, CD7/CD33, and HLA-DR/CD13 pairs of antibodies, where the 1st member of each pair was labeled with R-phycoerythrin and the 2nd with FITC.

=> s l15 and B cell associated antigen
   3 FILES SEARCHED...
L20   1 L15 AND B CELL ASSOCIATED ANTIGEN

=> d l20 cbib abs

L20 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
2001:538982 Document No.: PREV200100538982. Role of paraffin-section immunohistochemistry in the diagnosis of malignant lymphoma. Sadahira, Yoshito (1). (1) Department of Pathology, Kawasaki Medical School, Kurashiki, 701-0192 Japan. Kawasaki Medical Journal, (2000) Vol. 26, No. 2, pp. 83-98. print. ISSN: 0385-0234. Language: English. Summary Language: English.

AB Immunohistochemistry plays an integral role in the diagnosis of malignant lymphoma. The recent development of antibodies against fixative-resistant epitopes, the improvement of antigen retrieval techniques, and the introduction of highly sensitive detection systems have expanded the possibility of immunophenotyping of malignant lymphoma in routinely processed paraffin sections. This review provides precise information regarding antibodies useful in paraffin-section immunohistochemistry, as well as strategies for the immunohistochemical diagnosis of malignant lymphoma based on up-to-date classification.

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CA SUBSCRIBER PRICE		

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